

tion with the Children's Hospital comprises a national center for the study of lead toxicity of children supported by the National Institutes of Environmental Health Sciences, with additional support from the lead industry.

Dr. Kehoe conducted a study over a period of 34 years of the absorption, metabolism and excretion of lead in humans. It is the only study of its kind in the toxicology of lead. Human subjects were exposed to low levels of lead—via ingestion and others by inhalation. It should be noted that all of the subjects lived active, normal lives after their participation in the study. All of them are still living; most are now in their 70s and 80s. This work was published as a monograph in *Food and Chemical Toxicology* in June 1987.⁵

Dr. Robert A. Kehoe is now 96 years old and would probably be as unhappy about these omissions as I am. A historian of science should recognize that in the twenties and thirties there was little or no federal support for toxicological research at universities. The support from the private sector, including automotive, petroleum and even the lead industries, was welcomed by scientists such as Robert Kehoe.

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Response from Mr. Rabin

Dr. Suskind's letter misses the point of my references to scientists who were associated with the lead industry.¹ The purpose of identifying those researchers was not to discuss their accomplishments or to consider the effect of industry funding on their research.

The purpose of describing the ties of Robert Kehoe and other scientists to the lead industry was simply to make clear that what such researchers knew could not have been unknown to the Lead Industries Association or its members.

When Dr. Kehoe stated that "... strenuous efforts must be devoted to eliminating lead from [children's] environment,"² the lead pigment manufacturers could not claim that they were unaware of the dangers of lead paint to young children. Indeed, one of the reasons that one can refer to the "procrastination" of the paint industry in removing lead from paint is that industry-funded researchers were well aware of the dangers of lead paint long before the industry took action.

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On Borderline Statistical Significance

The title of the recent article by Maclure, *et al*, "Elevated Blood Levels of Carcinogens in Passive Smokers,"¹ misrepresents the data presented by the authors. While the design of the study is commendable, the conclusions drawn are not supported by the data presented. The data on 4-aminobiphenyl-hemoglobin (4-ABP-Hb) adducts are highly variable; the three highest levels of 4-ABP-Hb adducts are found in subjects with no detectable urinary cotinine. Additionally, the difference between cotinine-positive and cotinine-negative subjects is not statistically significant if the commonly accepted criterion of $p < 0.05$ is used. Additionally, it does not appear that 3-aminobiphenyl-hemoglobin (3-ABP-Hb) adducts can be considered a consistent marker of 4-ABP exposure as the subject with the highest level of 4-ABP adducts had no detectable level of 3-ABP adducts.

The changes in 3-ABP adduct levels between cotinine-positive and cotinine-negative subjects may have been statistically significant, but the large number of values below the limit of

detection and the low quantitative values of these adducts make the biological significance of the difference unclear. More importantly, there is little, if any, definitive evidence that 3-ABP is carcinogenic. The reference cited by the authors in support of this claim is a review article published in 1966² in which no data are presented nor citation provided on 3-ABP carcinogenicity. Furthermore, the authors, in a recent paper³ state that 3-ABP is reported to be noncarcinogenic. The rationale for studying 3-ABP-Hb adducts therefore appears to be lacking.

The variability and small differences among groups could be explained by non-tobacco sources of 4-ABP and 3-ABP, a possibility raised by these authors in a previous study.⁴ This previous study reported as much as a 300-fold difference among four animal species in their background levels of 4-ABP-Hb adducts (their Table 1), supporting the probability that, in the present study, these adducts may arise from non-tobacco sources.

While it is important to accurately assess the carcinogenic risk of passive exposure to tobacco smoke, this goal is not advanced when data are overinterpreted and a provocative, yet unsupported title is used. Unfortunately, this paper may be cited in the future as supportive of a cause and effect relationship which is not scientifically justified by the data presented.

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Response from Maclure, *et al*.

The difference of opinion between Reasor and us¹ concerns how to de-